

# Cardiac tamponade due to leukaemic pericardial effusion

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**Chia, B. L., Da Costa, J. L., and Ransome, G. A. (1973). *Thorax*, 28, 657–659. Cardiac tamponade due to leukaemic pericardial effusion.** A 19-year-old Chinese woman was admitted to hospital with cardiac tamponade. Pericardial fluid analysis and other investigations failed to reveal a cause of the pericardial effusion and she was treated as for tuberculous pericarditis. Subsequently haematological investigations showed that she was suffering from an acute lymphoblastic leukaemia.

Although cardiac involvement is seen at necropsy in approximately 20% of patients with malignant lymphomas or leukaemias, clinical evidence of these metastases during life is rare (Terry and Kligerman, 1970). There have been isolated reports of pericardial effusions developing in patients under treatment for leukaemia (Bierman, Perkins, and Ortega, 1952; Terry and Kligerman, 1970). However, it is very unusual for acute leukaemia to present as cardiac tamponade without any clinical or haematological evidence of the disease. To the best of our knowledge only one previous such case has been documented in the English medical literature (Wendkos, 1941).

In this paper we describe a patient with acute lymphoblastic leukaemia showing this most unusual mode of clinical presentation.

## CASE REPORT

A 19-year-old Chinese girl was first seen on 6 June 1970 with a one-week history of ankle oedema, exertional dyspnoea, and oliguria. Clinical examination revealed signs of a large pericardial effusion. The blood pressure was 100/70 mmHg and the pulse rate was 96 per minute. Pulsus paradoxus was present. No other abnormalities were detected clinically. A radiograph of the chest (Fig. 1) showed a large cardiac silhouette and bilateral pleural effusions. The electrocardiogram showed QRS complexes of very low voltage. Chemical and microscopic examination of the urine, blood urea, and serum electrolytes were all normal. The haemoglobin level was 15.2 g/100 ml (Haldane) and the total leucocyte count was 9,400/mm<sup>3</sup>—polymorphs 78%, lymphocytes 18%, mono-

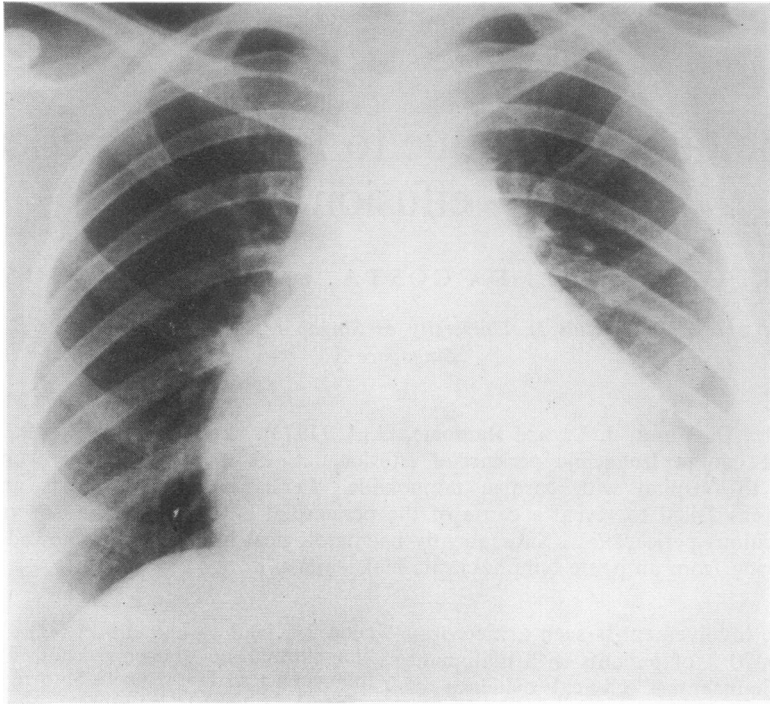
cytes 3%, and eosinophils 1%. The peripheral blood film showed no abnormal cells. The LE cell and tuberculin tests (1 TU) were both repeatedly negative.

Three hundred millilitres of clear yellowish coloured fluid was obtained from a pericardial aspiration. Microscopical examination of the pericardial fluid showed many lymphocytes but no abnormal cells nor acid-fast bacilli. The pericardial fluid was sent for tubercle bacilli culture and guinea-pig inoculation, both of which later proved to be negative. A radiograph of the chest taken after pericardial aspiration and injection of air showed a thickened parietal pericardium (Fig. 2).

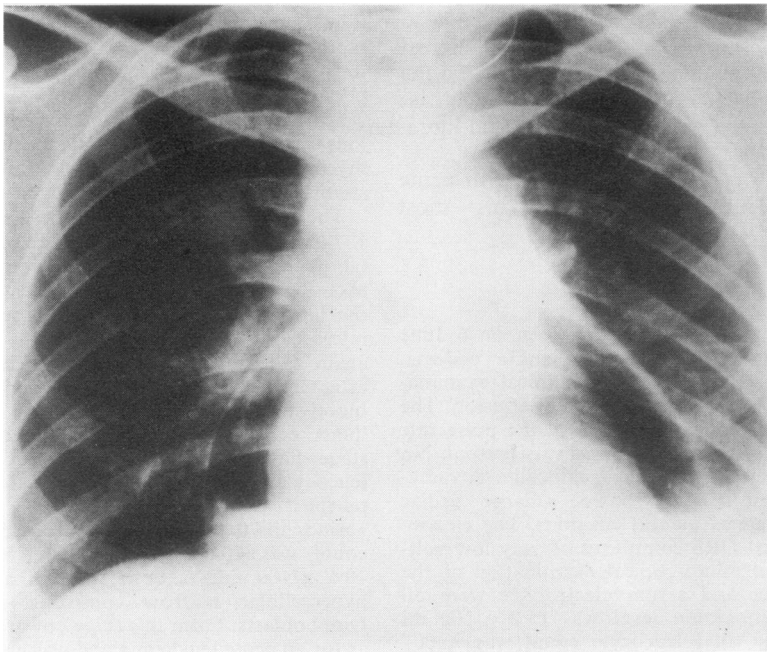
It was decided to treat the patient as for a tuberculous pericardial effusion and she was given streptomycin, oral para-amino salicylic acid, isoniazid, and steroids. Diuretics were also added to the therapy.

The patient was reasonably well until early August 1970 when she developed further episodes of cardiac tamponade which required paracentesis about once each week, increasing to once every other day during the later stages of her illness. Repeated cultures and microscopic examination of the pericardial fluid again failed to reveal any tubercle bacilli. On and after 29 September 1970, the pericardial fluid was blood-stained and for the first time showed numerous 'blast cells' on microscopic examination. At this stage haematological examination showed that the leucocyte count had increased to 58,000/mm<sup>3</sup>—polymorphs 41%, lymphocytes 4%, monocytes 1%, eosinophils 0%, and lymphoblasts 54%. The platelet count was 90,000/mm<sup>3</sup> and the haemoglobin level 10.6 g/100 ml. A bone marrow aspirate showed a hypercellular marrow consisting predominantly of lymphoblasts. From this stage onwards, she was treated as for an acute leukaemia and was given oral 6-mercaptopurine (150 mg daily) and prednisolone (120 mg daily). Nitrogen mustard (mustine) 5 mg, diluted in

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**FIG. 1.** *Radiograph of chest showing large cardiac silhouette.*



**FIG. 2.** *Radiograph of chest after pericardiocentesis and injection of air showing thickened parietal pericardium.*

20 ml of sterile water, was instilled into the pericardial cavity on one occasion.

During the subsequent month the episodes of cardiac tamponade became less frequent. However, the patient developed severe pancytopenia, her condition deteriorated rapidly, and terminally she was noticed to be deeply jaundiced. At this stage the serum bilirubin was 29.8 mg/100 ml, albumin 2.8 g/100 ml, globulin 2.0 g/100 ml, alkaline phosphatase 39.6 units, and serum glutamopyruvic transaminase 380 units. She died on 28 October 1970, four and a half months after the start of her illness. At no time was there any lymphadenopathy or splenomegaly.

#### NECROPSY FINDINGS

The whole pericardium was found to be thickened irregularly with solid tumour masses. The myocardium in both the ventricles and the auricles was diffusely infiltrated with streaks of tumour tissue. A solid tumour mass, measuring 4 by 4 cm, was seen in the anterior mediastinum.

Histological examination of the pericardium, myocardium, and the anterior mediastinal mass revealed a uniform cellular infiltration of abnormal 'blast' cells.

#### DISCUSSION

In 1941 Wendkos described a case of lymphatic leukaemia presenting with cardiac tamponade. Although the pericardial fluid failed to reveal

tubercle bacilli on both microscopy and guinea-pig inoculation, a diagnosis of tuberculous pericarditis was made. Subsequently her condition deteriorated, and about two months after the start of her illness haematological investigations revealed lymphatic leukaemia. The patient reported in the present paper followed a similar course.

In 1967 Rab and Yee described a patient with acute leukaemia presenting with cardiac tamponade. However, unlike the patient described in this paper and the one reported by Wendkos (1941), the diagnosis of acute leukaemia was evident from routine haematological investigations at the onset of the illness.

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